SKIN & KIDNEY

BY

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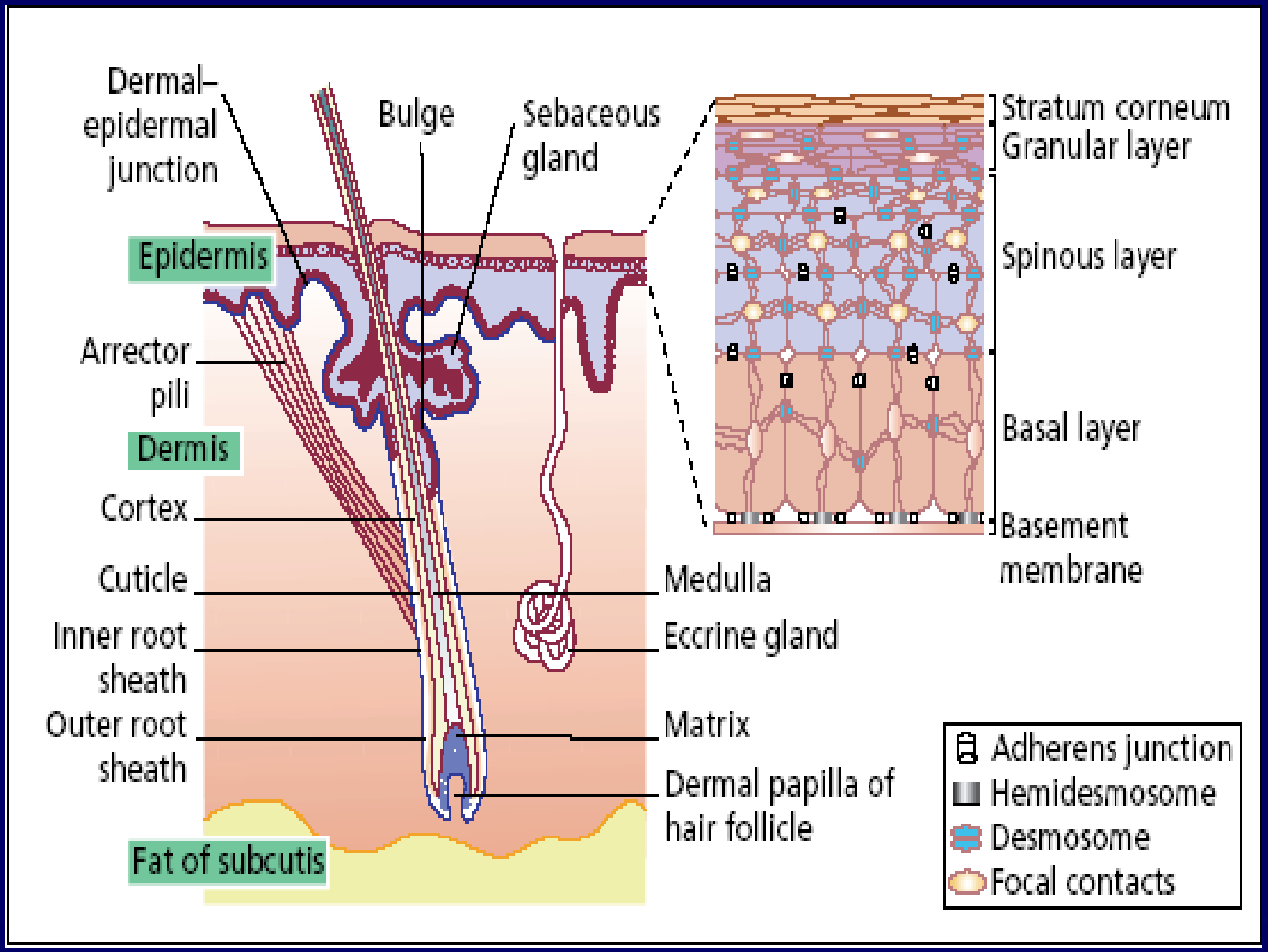
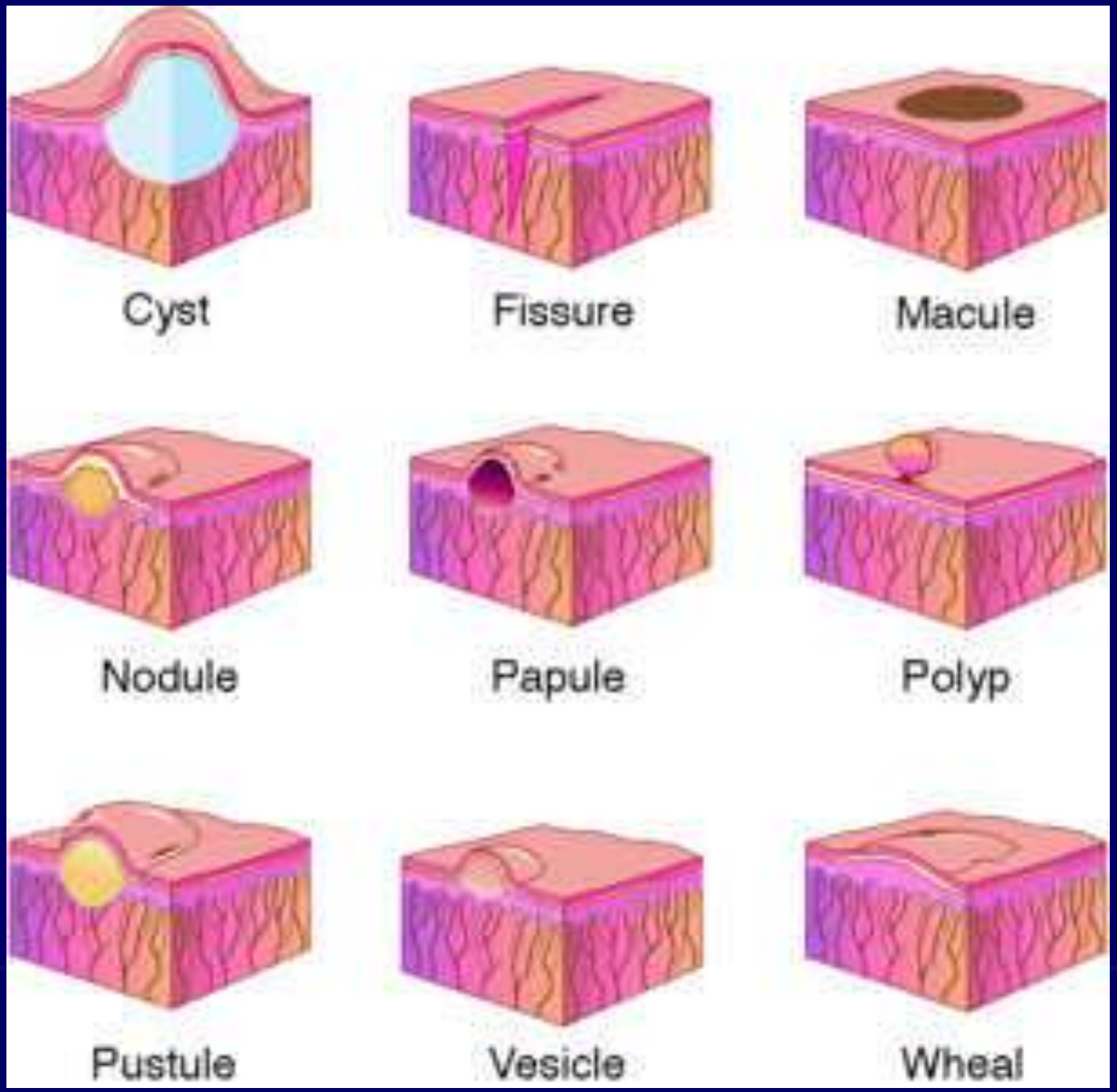
كاريكاتير لكارلوس لانوف 8-7-2012



THE SKIN AND ITS APPENDAGES.


Cyst
Fissure
Macule
Nodule
Papule
Polyp
Pustule
Vesicle
Wheal


شهداء ثورة 25 يناير
ولا تحسبن الذين قتلوا فى سبيل الله امواتا بل احياء عند ربهم يرزقون

MULTISYSTEM DISORDERS

RELATED TO DIALYSIS

Related to transplantation

END STAGE RENAL DISEASE (ESRD)

MULTISYSTEM DISORDERS

- **SLE**
- Vasculitis : **cryoglobulonemia**, polyarteritis nodosa, **HSP**
- **Streptococcal infection**
- **Fabry's disease**
- Scleroderma
- Tuberous scleroses
- Neurofibromatosis
- 1ry systemic amyloidosis

ANGIOKERATOMA CORPORIS DIFFUSUM (FABRY'S DISEASE)

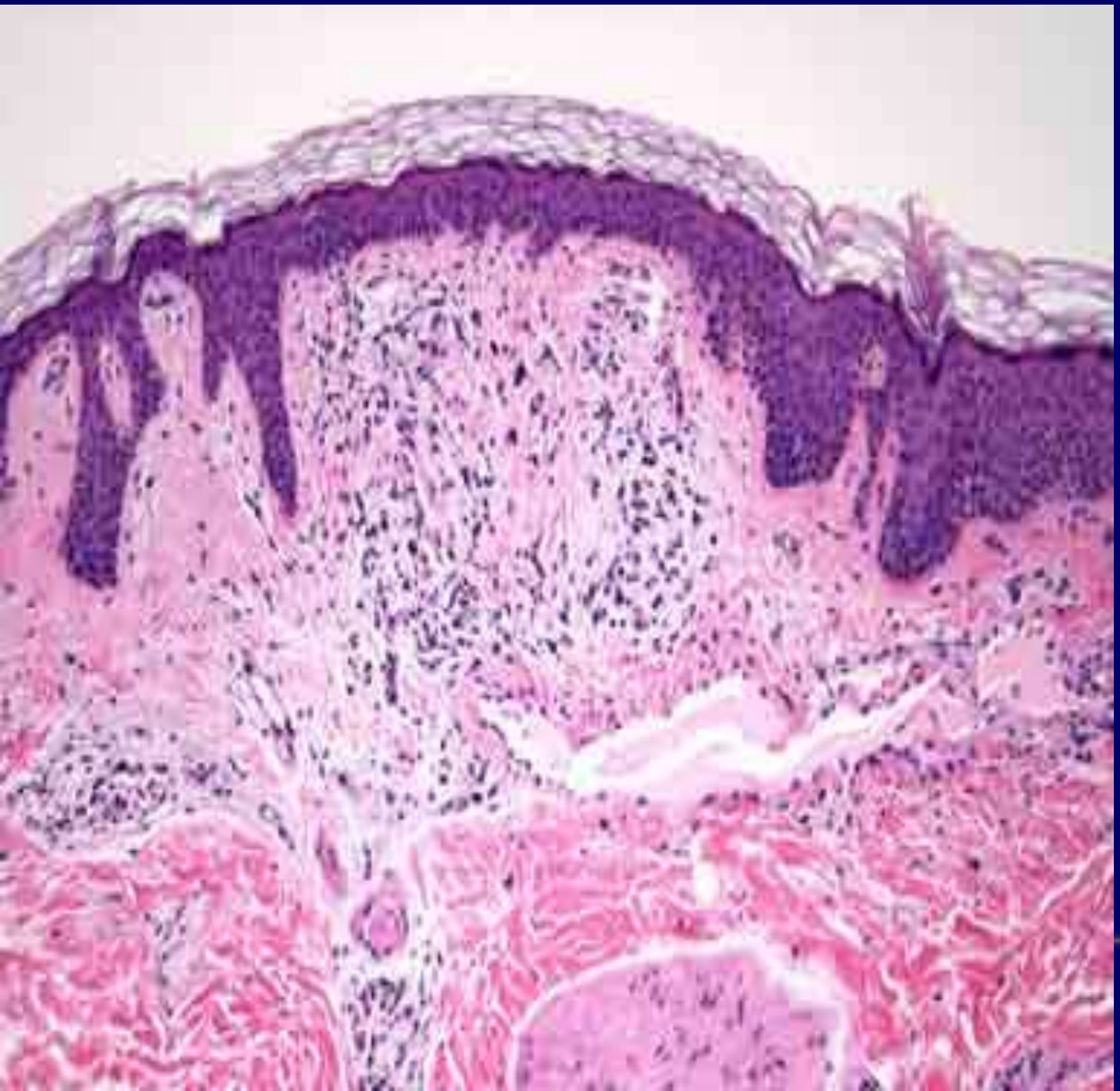
ANGIOKERATOMA CORPORIS DIFFUSUM (FABRY'S DISEASE)

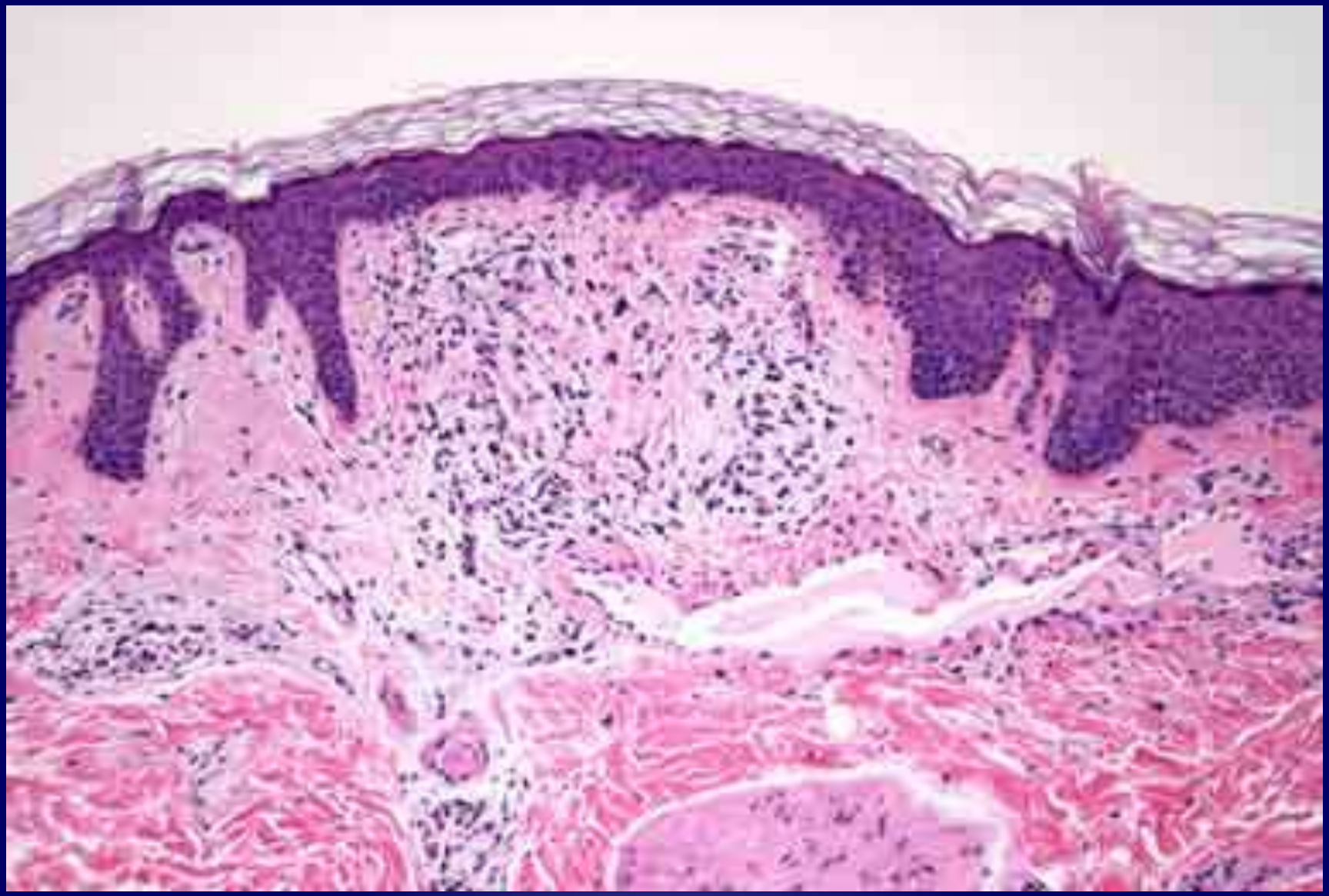
- A 25-year-old male
- Presented with hypertensive encephalopathy
- Examination revealed uraemic manifestations with characteristic skin eruption
- Clinicopathological diagnosis: Angiokeratoma corporis diffusum

ANGIOKERATOMA CORPORIUM DIFFUSUM (FABRY'S DISEASE)

- X-linked recessive
- Deficiency of alpha-galactosidase A progressive accumulations of ceramide trihexoside in most visceral tissues
- Numerous small angiokeratoma between the umbilicus and the knee, dry skin & anhidrosis
- Renal insufficiency, HTN, paresthesia heart and CNS affection
- Death usually occurs from cardiac or renal failure
- TTT: renal transplant


http://derm.net





Ectatic blood vessels in papillary dermis with overlying epidermal hyperplasia & hyperkeratosis

Cytoplasmic vacuoles representing lipids can sometimes be detected in endothelial cells, fibroblasts and pericytes

SLE

ARA CRITERIA FOR DIAGNOSIS OF SLE

(PRESENCE OF ANY 4 OR MORE OF 11 SERIALLLY OR SIMULTANEOUSLY IS DIAGNOSTIC)

- Malar rash
- Discoid rash
- Photosenstivity
- Oral ulcers
- Non-rosive arthiritis
- Seositis-pleuritis or pericarditis
- Renal disorders: persistant protinuria (more than 500 mg/day) or cellular casts
- Neurological disorders: seizzures or psychosis
- Hematological disorders: hemolytic anaemia or leukopenia or lymphopenia or thrombocytopenia
- Immunological: LE cell or anti-nDNA Abs or anti-SmAbsor false +ve serological tests for $ (more than 6 months)
- ANA

LUPUS NEPHRITIS

- Usually arises within 5 years of diagnosis
- Symptoms are generally related to hypertension, proteinuria, and renal failure.
- Evaluating renal function in patients with SLE to detect any renal involvement early is important because early detection and treatment can significantly improve renal outcome.
- Renal biopsy should be considered in any patient with SLE who has clinical or laboratory evidence of active nephritis, especially upon the first episode of nephritis.
- Lupus nephritis is staged according to the classification revised by the International Society of Nephrology (ISN) and the Renal Pathology Society (RPS) in 2003. This classification is based on light microscopy, immunofluorescence, and electron microscopy findings from renal biopsy specimens.

IMPETIGO CONTAGIOSA

- Caused by nephrogenic streptococcal strains
- Subacute glomerulonephritis
- Early ttt is mandatory

HENOCH SCHONLEIN PURPURA

PATHOPHYSIOLOGY

- **Henoch-Schonlein purpura** (HSP):
- Small-vessel vasculitis
- Immunoglobulin A (IgA), C3, and immune complex deposition in arterioles, capillaries, and venules.
- HSP and IgA nephropathy are related disorders.
- Both illnesses have elevated serum IgA levels and identical findings on renal biopsy
- However, IgA nephropathy almost exclusively involves young adults and predominantly affects the kidneys only.
- HSP affects mostly children and involves the skin and connective tissues, gastrointestinal tract, joints, and scrotum as well as the kidneys.[2, 3, 4]

PHYSICAL

Palpable purpura • Typical distribuation •

HSP

HCP

THERAPY

- Very limited data are available
- Fortunately, most patients recover quickly in several weeks without treatment.[5]
- NSAIDs may help joint pain
- However, NSAIDs should be used cautiously in patients with renal insufficiency.

- To use corticosteroids or not to use conflecting data

- Prednisone in a dose of 1 mg/kg/d for 2 weeks and then tapered over 2 more weeks has been shown to improve gastrointestinal and joint symptoms.[11]
- Other treatment regimens included IV or oral steroids with or without any of the following:
  - azathioprine,
  - cyclophosphamide,
  - cyclosporine,
  - dipyridamole,
  - plasmapheresis,
  - high-dose IV immunoglobulin G (IVIg),
  - danazol, or fish oil.

- One study of 12 patients with severe Henoch-Schönlein purpura (HSP) nephritis indicated that patients did well with a treatment of methylprednisolone at 30 mg/kg/d for 3 days followed by oral corticosteroids at 2 mg/kg/d for 2 months, cyclophosphamide at 2 mg/kg/d for 2 months, and dipyridamole at 5 mg/kg/d for 6 months.

PROGNOSIS

- Generally a benign disease with an excellent prognosis.
- More than 80% of patients have a single isolated episode lasting a few weeks.
- 10-20% of patients have recurrences.
- Fewer than 5% of patients develop chronic Henoch-Schönlein purpura.
- Abdominal pain resolves spontaneously within 72 hours in most patients.

CRYOGLOBULINEMIA

- Single or mixed immunoglobulins that undergo reversible precipitation at low temperatures

- **Three types:** Type I (monoclonal) Type II and Type III(Mixed) cryoglobulins

- **Type I cryoglobulins:**
  - monoclonal Ig(IgG or IgM, although IgA and Bence Jones proteins)
  - may present with occlusive vasculopathy or may not exhibit any specific clinical manifestations.

- **Mixed (type II and type III) cryoglobulins:**

  immune complexes consisting of a rheumatoid factor (RF, the antibody), usually IgM, complexed with IgG (the antigen).
  - Type II cryoglobulins:monoclonal Ig (usually IgM)+ polyclonal IgG.
  - Type III cryoglobulins:polyclonal Ig + polyclonal IgG or a non Ig serum component.
  - Both types usually occur with CTD, infection, hepatic disease, or lymphoproliferative disease.
  - depressed levels of C3, along with decreased levels of C2, and C4

- Type II
  - Clinical presentation of
    - Cutaneous vasculitis-purpura/leg ulcers
    - Arthralgias/arthritis
    - Immune-complex nephritis
    - Neuropathy
  - Renal disease characterized histologically by
    - Membranoproliferative glomerulonephritis
    - Hyaline inclusions in glomeruli, positive for immunoglobulin M
    - Vasculitis
    - Congophilic-negative fibrillar inclusions in glomeruli by electron microscopy
  - Sjögren syndrome complicated by nephritis, glomerulonephritis, or gammopathy
  - Chronic hepatitis with extra-hepatic clinical manifestations
  - High-titer rheumatoid factor activity in the absence of rheumatoid disease
  - Markedly depressed levels of C4

RELATED TO DIALYSIS:

- Pruritis
- Splinter hemorrahage
- Bullous dermatosis of hemodialysis
- Uremic neuropathy
- Gynecomastia
- Pseudo PCT
- Acquired perforating dermatosis

RELATED TO TRANSPLANTATION

- Increased incidence of infections: fungal, bacterial, norwagian scabies, atypical warts
- Cushing's syndrome
- Increase of cancers: SCC, kaposi sarcoma

Kaposi sarcoma

Bacillary angiomatsis

END STAGE RENAL DISEASE (ESRD)

CUTANEOUS MANIFESTATIONS OF END-STAGE RENAL DISEASE ESRD

Non Specific disorders:

- **Skin color changes**
  - **Pallor**
  - **Sallow yellowish cast**
  - **Hyperpigmentation**
  - **Elastosis**
  - **Ecchymoses**
- **Xerosis, poor skin turgor, acquired ichthyosis**
- **Uremic frost**
- **Half-and-half nails**
- **Pruritus**

Specific disorders

- **Perforating disorders**
- **Metastatic calcification**
  - **Benign nodular calcification**
  - **Calciphylaxis**
- **Bullous dermatosis**
  - **Porphyria cutanea tarda**
  - **Pseudoporphyria**
- **Fibrotic uremic dermopathy (FUD), nephrogenic systemic fibrosis (NSF).**

CHANGES IN SKIN COLOR

Pallor:

- Results from anemia caused by chronic disease.

A sallow yellowish cast to the skin:

- From urochrome and carotinoid deposition.

Hyperpigmentation:

- Photodistributed.

CHANGES IN SKIN COLOR

Elastosis :

- Often distributed in sun-exposed areas.
- Yellowish plaques
- Histologically shows basophilic degeneration of collagen in the upper dermis.
- The relationship of elastosis to ESRD is not clear.

Ecchymoses:

- Secondary to platelet dysfunction.

Spontaneous ecchymotic injury on the upper limb of a patient with chronic renal failure

HYPERCHROMIC MACULES WITH A RETICULATED ASPECT IN THE FRONTAL REGION OF A PATIENT WITH CHRONIC RENAL FAILURE UNDERGOING HEMODIALYSIS

Xerosis → ichthyosiform scaling.

Uremic frost:

- **Now rarely seen.**
- **Superficial white deposits that are secondary to crystallized urea excreted from sweat.**

SKIN XEROSIS EVOLVING WITH ICHTHYOSIFORM APPEARANCE IN THE LOWER LIMB OF A CHRONIC RENAL PATIENT

Half-and-half nails (Lindsay's nails):

- In one third of patients with azotemia.
- The proximal half of the nail appears white because of edema of the nail bed and capillary network.
- The distal portion appears normal or brown.
- The nail plate is unaffected.
- There is no correlation with the severity of uremia.

HALF AND HALF NAIL

PRURITUS

- The most frequent symptom of ESRD,
- 58% to 90% of patients undergoing maintenance dialysis.
- The prevalence is similar in patients treated with hemodialysis and in patients on continuous ambulatory peritoneal dialysis.
- localized or generalized.
- Often with intermittent intense paroxysms that disrupt sleep and daily activities.

CLINICAL FEATURES OF PRURITUS

- Excoriations.
- Lesions of lichen simplex chronicus.
- Prurigo nodularis.
- Pruritus also contributes to koebnerization of acquired perforating dermatosis.

HISTOLOGIC FEATURES

- Atrophy of adnexal structures including sebaceous glands.
- Microangiopathy with:
  - *Endothelial cell activation or necrosis,*
  - *Basement membrane zone thickening,*
  - *Reduplication of the basal lamina of venuoles and arterioles.*
- Mast cell concentration varies.
- Slight spongiosis and hyperkeratosis without parakeratosis.

PATHOPHYSIOLOGY

- Unknown ⟶ multifactorial.
- A variety of theories have been proposed:
  - Xerotic skin.
  - Hypervitaminosis A
  - Urochromes retention
  - Uremic toxins retention
  - Elevated PTH, calcium, phosphate, and magnesium levels,
  - Increased mast cells in the skin of patients with ESRD
  - Abnormal pattern of cutaneous innervation

MANAGEMENT

Curative therapy for uremic pruritus is successful kidney transplantation.

OTHER LINES OF THERAPY

1.UVB phototherapy:

- **The most effective therapy.**
- **The mechanism of action:**
  - **Photoinactivation of pruritogenic substances,**
  - **Formation of photoproducts with antipruritic effects,**
  - **Production of vitamin D leading to alteration of divalent ions.**

OTHER LINES OF THERAPY (CONT.)

2. **Oral activated charcoal.**
3. **Naltrexone and nalaxone. (opioid mu receptor antagonists).**
4. **Numerous other therapies with variable success and length of remissions.**
   - **Soaks, baths, lubrication,**
   - **Parathyroidectomy,**
   - **Antihistamines,**
   - **Cholestyramine**
   - **Intravenous lidocaine,**
   - **Nicergoline,**
   - **Neurotropin,**
   - **Thalidomide,**
   - **Acupuncture**

PERFORATING DISORDERS IN ESRD

(ACQUIRED PERFORATING DERMATOSIS, APD)

features of classic KD, PF, and RPC:

- Keratotic papules, nodules, and occasionally verrucous plaques on hair-bearing areas subject to friction.
- Trunk and extremities >face and scalp.
- Papules are often umbilicated with a central keratotic plug.
- Follicular and perifollicular
- Koebnerization may be observed,
- Pruritus is usually severe
- Spontaneous resolution of individual lesions with the continued development of new lesions.

HISTOLOGY OF APD

May resemble KD, PF, or RPC, or combine features of these perforating disorders.

PATHOPHYSIOLOGY OF APD

Transepidermal elimination:

- An abnormality of epidermal proliferation or,
- Vasculopathy and pruritus ⟹ alteration of dermal connective tissue.
- Elimination of the altered connective tissue.

Leukocytes may play a role in the pathogenesis:

- In transepidermal elimination, granular-globular DNA material is eliminated corresponding to nuclear debris of necrotic leukocytes.
- Collagenase and elastase released from disintegrating leukocytes may also alter dermal connective tissue.
- Ieukocyte proteinases may also act to loosen keratinocyte intercellular bridges, facilitating the extrusion of this material through the epidermis.

DIFFERENTIAL DIAGNOSIS OF APD

- primary perforating disorders, KD, PF, and RPC.
- prurigo nodularis,
- verruca vulgaris,
- eruptive keratoacanthomas,
- phrynoderma,
- keratotis pilaris,
- hypertrophic lichen planus.

MANAGEMENT OF APD

- **Potent topical steroids,**
- **Topical steroids under occlusion,**
- **Interlesional steroids**
- **Topical retinoids,**
- **Oral vitamin A (100,000 U/day)**
- **Oral retinoids**
- **Cryotherapy.**
- **Keratolytics**

APD. UMBILICATED PINK PAPULES WITH CENTRAL KERATOTIC PLUGS IN LIGHT SKIN.

CALCIFYING DISORDERS OF THE SKIN

- **Metastatic calcification is the precipitation of calcium salts in normal tissue due to defects in calcium or phosphate metabolism.**
- **In chronic renal failure, cutaneous metastatic calcification presents as:**
  - ***Benign nodular calcification (calcinosis cutis) or***
  - ***Calciphylaxis.***

CALCINOSIS CUTIS

Clinical features

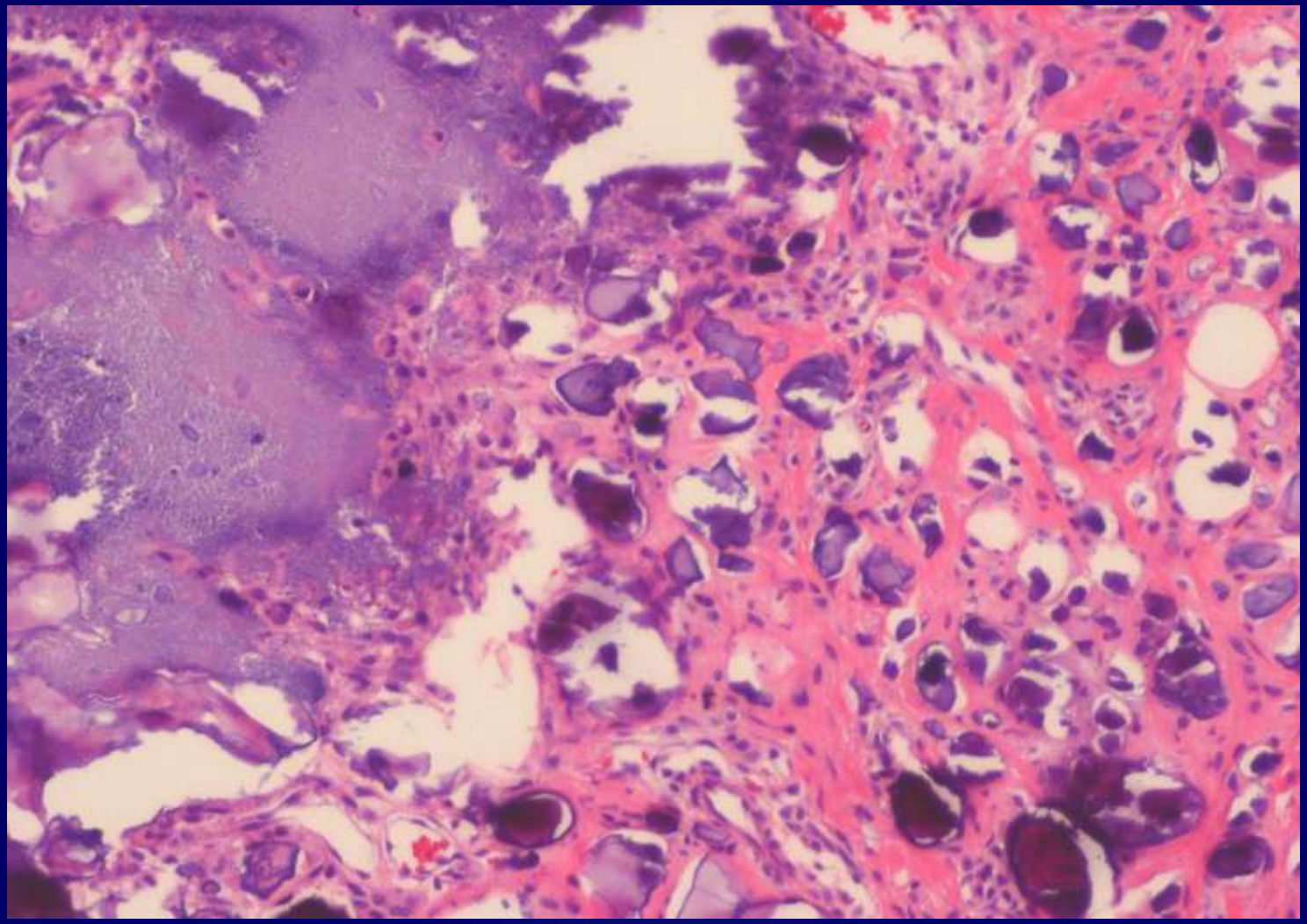
- **Firm papules, plaques, and nodules**
- **A chalky discharge may be extruded**
- **On periarticular sites (asymptomatic) and fingertips (painful).**
- **The degree of involvement correlates with calcium and phosphate levels.**

can disappear with normalization of the calcium and phosphate levels below the solubility product.

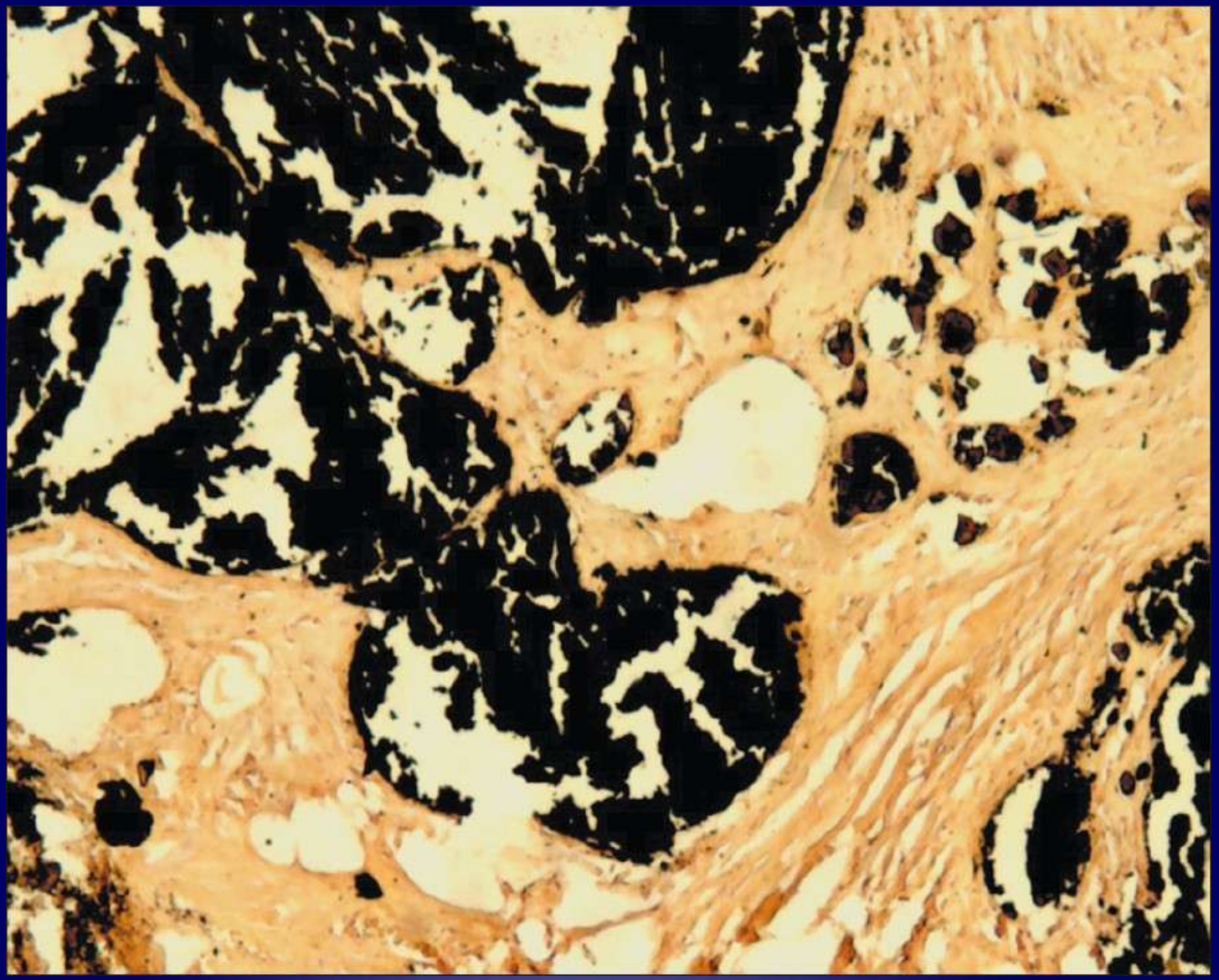
HISTOPATHOLOGY

- On hematoxylin-eosin:
  - Homogeneous blue material (calcium) in the superficial dermis or as deep globular deposits in the subcutaneous tissue.
  - Foreign body giant cells.

- Von Kossa stains the deposits black.



METASTATIC CALCINOSIS CUTIS. AMORPHOUS DEPOSITS OF BASOPHILIC MATERIAL ARE PRESENT IN THE DERMIS.



VON KOSSA STAINS THE DEPOSITS BLACK.

PATHOPHYSIOLOGY

- Reduced clearance of phosphate,
- Secondary hyperparathyroidism:

Due impaired production of vitamin D,

↓

lower intestinal calcium absorption

↓

decreased serum calcium levels.

↓

mobilization of calcium and phosphate from bone into serum.

MANAGEMENT

- phosphorus- binding antacids,
  - The recently approved organic phosphatge binder ***RenaGel*** (a polyallylamine hydrochloride) is now recommended as the antacid of choice.
- Parathyroidectomy.
- Diet low in phosphate

SO Avoid

- Milk and milk products;
- Vegetables such as artichokes, broccoli, brussel sprouts;
- High-protein foods such as oysters, flounder, liver, salmon;
- Beverages such as beer, ale, colas, cocoa; and
- Miscellaneous foods such as nuts, seeds, wheat germ, and caramels.

- الحليب ومنتجاته ،
- الخضراوات مثل الخرشوف ، القرنبيط ، وملفوف بروكسل ،
- الأطعمة عالية البروتين مثل المحار والسمك المفلطح ، والكبد ، سمك السلمون ،
- والمشروبات مثل البيرة ، والبيرة ، الكولا ، والكاكاو ،
- و الأطعمة المتنوعة مثل المكسرات والبذور وجنين القمح ، والكراميل.

BENIGN NODULAR CALCIFICATION (CALCINOSIS CUTIS). FIRM SUBCUTANEOUS NODULES ADJACENT TO THE ELBOW.

CALCIPHYLAXIS

- **The term calciphylaxis was coined by *Selye in 1962* as a condition of hypersensitivity in which sensitized tissues respond to appropriate challenging agents with calcium deposition**

CALCIPHYLAXIS

- A devastating (مدمر)
- Life-threatening condition of progressive cutaneous necrosis secondary to small vessel calcification.
- Incidence is 4% in dialysis patients and 1% in patients undergoing conservative treatment
- Mortality rates from 60% to 80%.
- Other causes:
    - Transplant receipient
    - Primary hyperparathyroidism,
    - Crohn's disease,
    - AIDS, and
    - Cirrhosis

CALCIPHYLAXIS RISK FACTORS

Implicated sensitizing agents

- Elevated PTH levels
- Elevated calcium × phosphate product: 70 or higher mg^2/dl^2 are at great risk
  - 2 ([C][P] – 5) × (alkaline phosphatase) × (PTH ratio): 1000 U or higher
  - *The PTH ratio is defined as the ratio of the patient's PTH level divided by normal PTH levels.*
- Vitamin D

Suspected challenging agents

- Albumin
- Metallic salts
- Calcitriol
- Corticosteroids
- Local trauma
- Lymphoma
- Immunosuppressants
- HIV

CLINICAL FEATURES

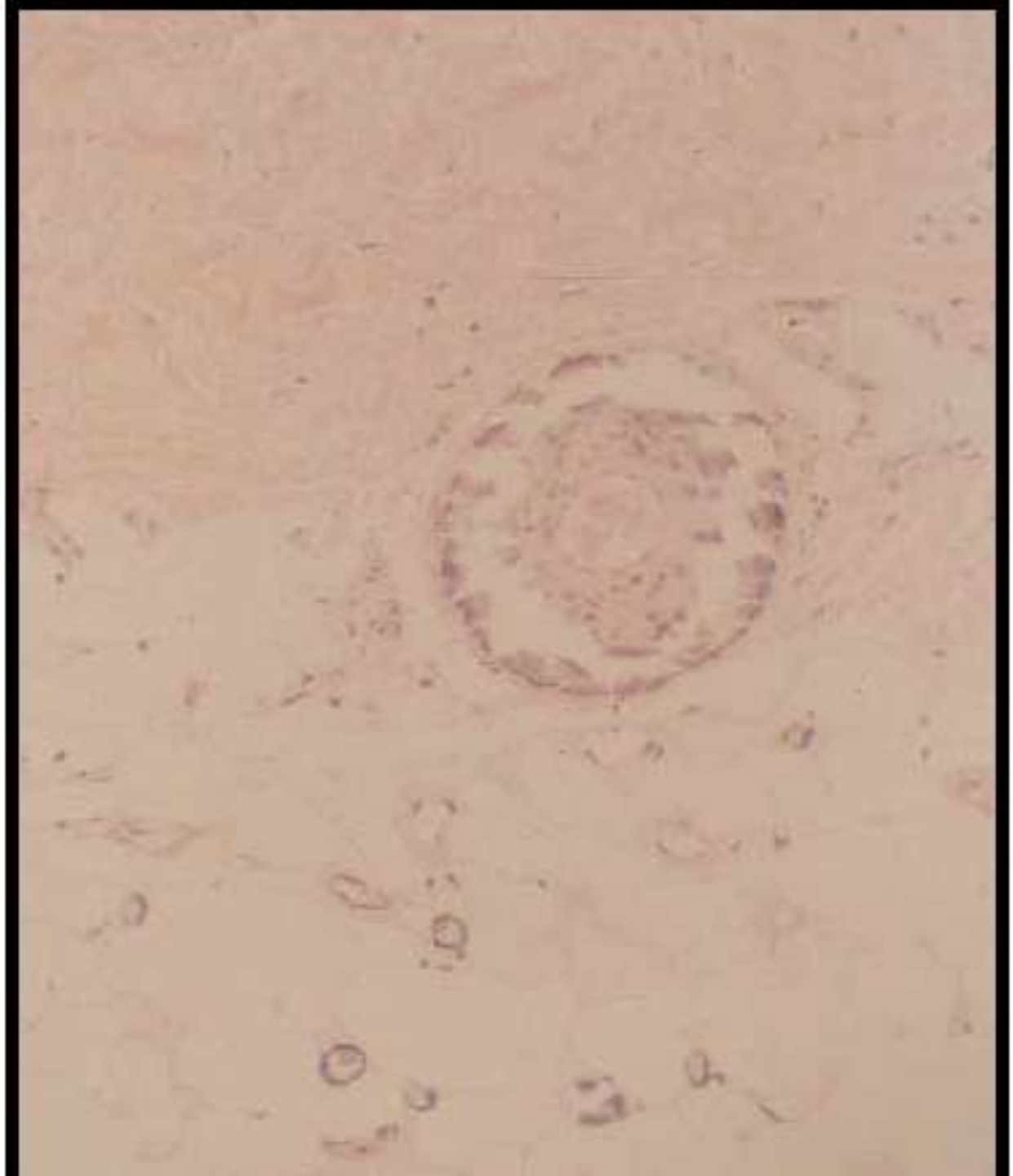
- **Plaques or nodules:**
  - Firm,
  - bilateral, symmetric,
  - painful
  - purpuric
  - with a reticulated pattern (retiform purpura)
  - eventuating in superficial nor deep angulate or stellate ulcers with eschar or frank gangrene.
- Acral distribution have a better prognosis than proximal distribution.
- Peripheral pulses are preserved distal to the area of necrosis.
- Myopathy, hypotension, fever, dementia, and infarction of CNS, bowel, or myocardium.

HISTOPATHOLOGY

- Medial calcification and intimal hyperplasia of small arteries and arterioles of dermal and subcutaneous tissue
- Fibrin thrombi
- Epidermis: ischemic necrosis.
- Delicate calcium deposition surrounding lipocytes or global calcification of septal capillaries in subcutaneous tissue.
- Panniculitis with lobular fat necrosis and mixed inflammation composed of neutrophils, lymphocytes, and histiocytes with rare septal involvement

DIAGNOSIS

- **Radiographic findings:**
  - Plain x ray(not specific)
    - medial calcification is recognizable as a fine double-lined network.
    - small vessel with a diameter less than 0.5 mm
  - Mammography:
    - The most sensitive radiologic detection.
  - Duplex sonography.
  - Angiography if necessary.
- **Incisional biopsy The most useful technique for diagnosis**



HISTOPATHOLOGIC FEATURES OF CALCIPHYLAXIS. MEDIAL CALCIFICATION AND INTIMAL HYPERPLASIA OF AN ARTERIOLE AT THE DERMAL-SUBCUTANEOUS JUNCTION. NOTE CALCIFICATION OF INTERLOBULAR CAPILLARIES IN THE SUBCUTANEOUS TISSUE.

Work-up of calciphylaxis

- Blood urea nitrogen/creatinine
- Calcium/phosphate levels
- PTH levels
- ANA, ANCA
- Cryoglobulins, cryofibrinogen
- Hepatitis B and hepatitis C profile
- Amylase
- X-rays
- Incisional skin biopsy for hematoxylin-eosin staining
- Culture and Sensitivity
- DIF
- Assessment of peripheral pulses
  - Oscillography
  - Transcutaneous oxygen pressure
  - Duplex sonography
  - Angiography (if necessary)

PROGNOSIS

- Poor with a 60% to 80% mortality rate from:
  - Sepsis,
  - Organ failure,
  - Inanition events.
- Proximal locations of necrosis carry an unfavorable prognosis compared with distal involvement.

MANAGEMENT

Prevention

- Reducing calcium and phosphate levels,
- Preventing secondary hyperpara-thyroidism,
- Minimizing obesity and local trauma.

CURATIVE MANAGEMENT

- Low phosphate diet (<43 mg/day),
- Low calcium dialysate.
- Dietary phosphate binders without calcium carbonate.
- Known triggers should be minimized, including obesity, injections into proximal adipose tissue, blood products, and immunosuppressants.
- Aggressive wound care:
  - Debridement, systemic antibiotics, hydrocolloid dressings, biologic dressings, or split-thickness grafts.
- Subtotal parathyroidectomy or total parathyroidectomy
  - With autotransplantation of one gland into the forearm
  - In patients with hyperparathyroidism.

Hyperbaric oxygen therapy:

- In patients undergoing both peritoneal and hemodialysis.
- Wounds that did not respond to medical management, parathyroidectomy, and aggressive debridement.

- **Promotes wound healing by:**
  - Elevating the partial pressure of oxygen within diseased tissue,
  - Improving angiogenesis and phagocytosis,
  - Inhibiting bacterial growth, decreasing local tissue edema.

CALCIPHYLAXIS

BULLOUS DERMATOSES IN ESRD
PORPHYRIA CUTANEA TARDA

- PCT is a disorder of hepatic heme biosynthesis associated with uroporphyrinogen decarboxylase deficiency
- The clinical presentation of PCT associated with ESRD does not differ from that observed in sporadic PCT.
  - tense vesicles, bullae, erosions and crusts on the dorsal hands, face, feet.
  - heal with scarring and milia formation.
  - The skin is fragile
  - Hyperpigmentation of sun-exposed
  - Hypertrichosis
  - Sclerodermoid plaques

LABORATORY FINDINGS

increased serum iron, ferritin, and hepatocellular iron.

Patients who are not anuric:

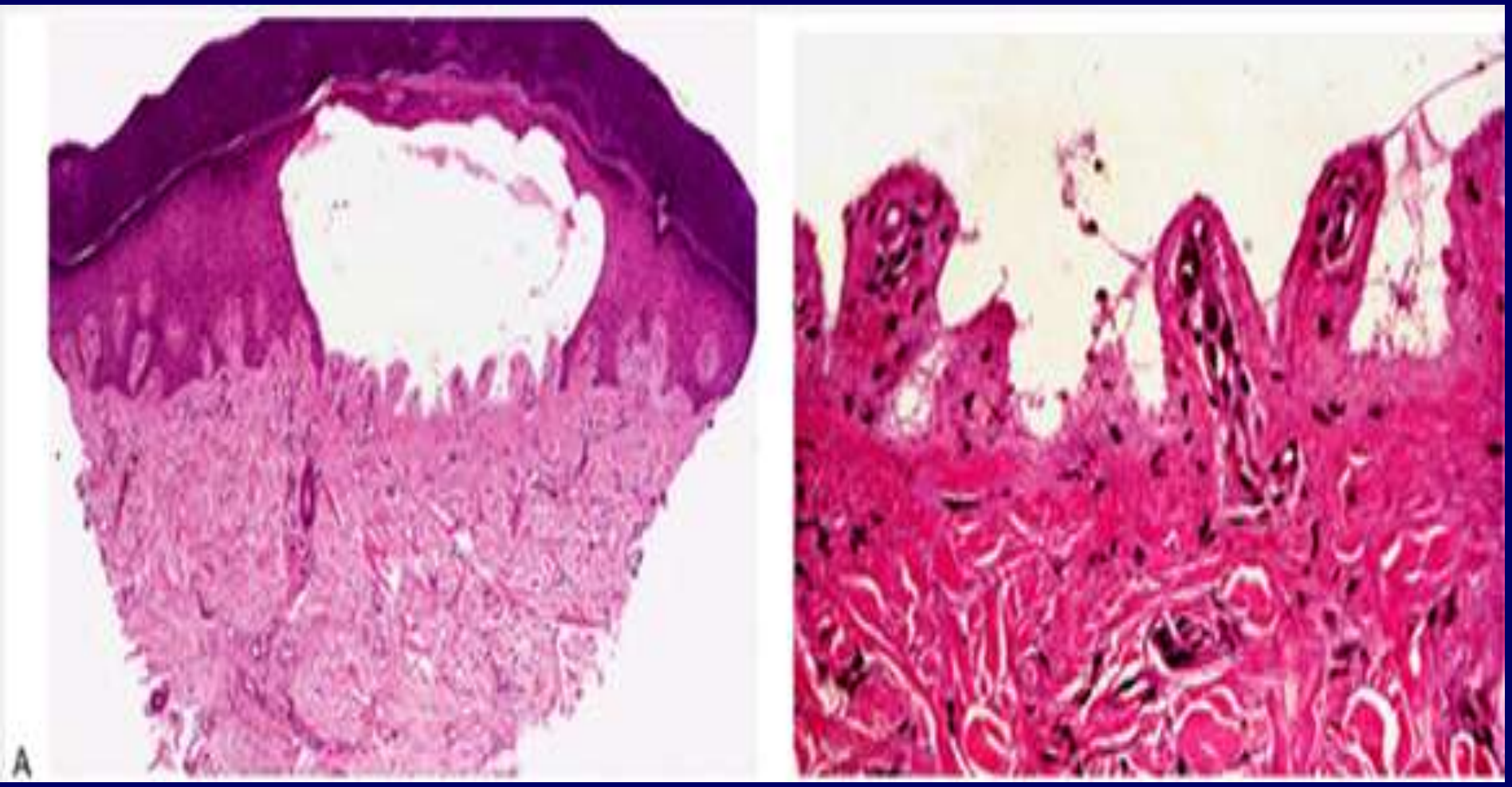
- **Urine:**
  - increased uroporphyrin I >uroporphyrin III, 8-carboxyl uroporphyrin, and 7-carboxyl porphyrins.
- **Feces:**
  - Isocoproporphyrin III levels are increased

Anuric patients with ESRD

- markedly elevated plasma levels of uroporphyrin.
- Standard hemodialysis does not effectively remove uroporphyrins.

HISTOPATHOLOGY

- Subepidermal vesiculation
- little or no inflammation.
- The base of the bulla shows festooning.
- mild thickening of the basement membrane zone and vessel walls, highlighted with PAS stain
- Intraepidermal eosinophilic wormlike or caterpillar bodies
- DIF of lesional skin: linear IgG, C3, and fibrin in a granular pattern at the DEJ and around blood



PORPHYRIA CUTANAE TARDA.A :THERE IS A SUBEPIDERMAL BLISTER. THE ARCHIECTURE OF THE DERMAL PAPILLAE REMAINS INTACT. THE DERMIS CONTAINS NO SIGNIFICANT INFLAMMATORY INFILTRATE (H&E)

D.D. OF PCT IN PATIENTS WITH ESRD

- Pseudoporphyria.
- Other forms of hereditary porphyria and hereditary coproporphyria,
- drug-related phototoxic reactions,
- primary subepidermal bullous diseases

MANAGEMENT

- Avoidance of environmental triggers such as alcohol and estrogens.
- A broad-spectrum physical blocker such as zinc oxide or titanium dioxide.
- High flow rate dialysis with a high-flux polysulfate dialyzer (standard hemodialysis does not effectively remove uroporphyrin.)

- Small volume phlebotomies (50-100 mL) every week over 1 year. (Patients with ESRD cannot tolerate standard phlebotomy of 500 mL every other week)
- Erythropoietin in conjunction with support phlebotomies
- Iron chelators, such as deferoxamine,
- Chloroquine is not effective in anuric patients.
- Plasmapheresis and plasma exchange with limited success.

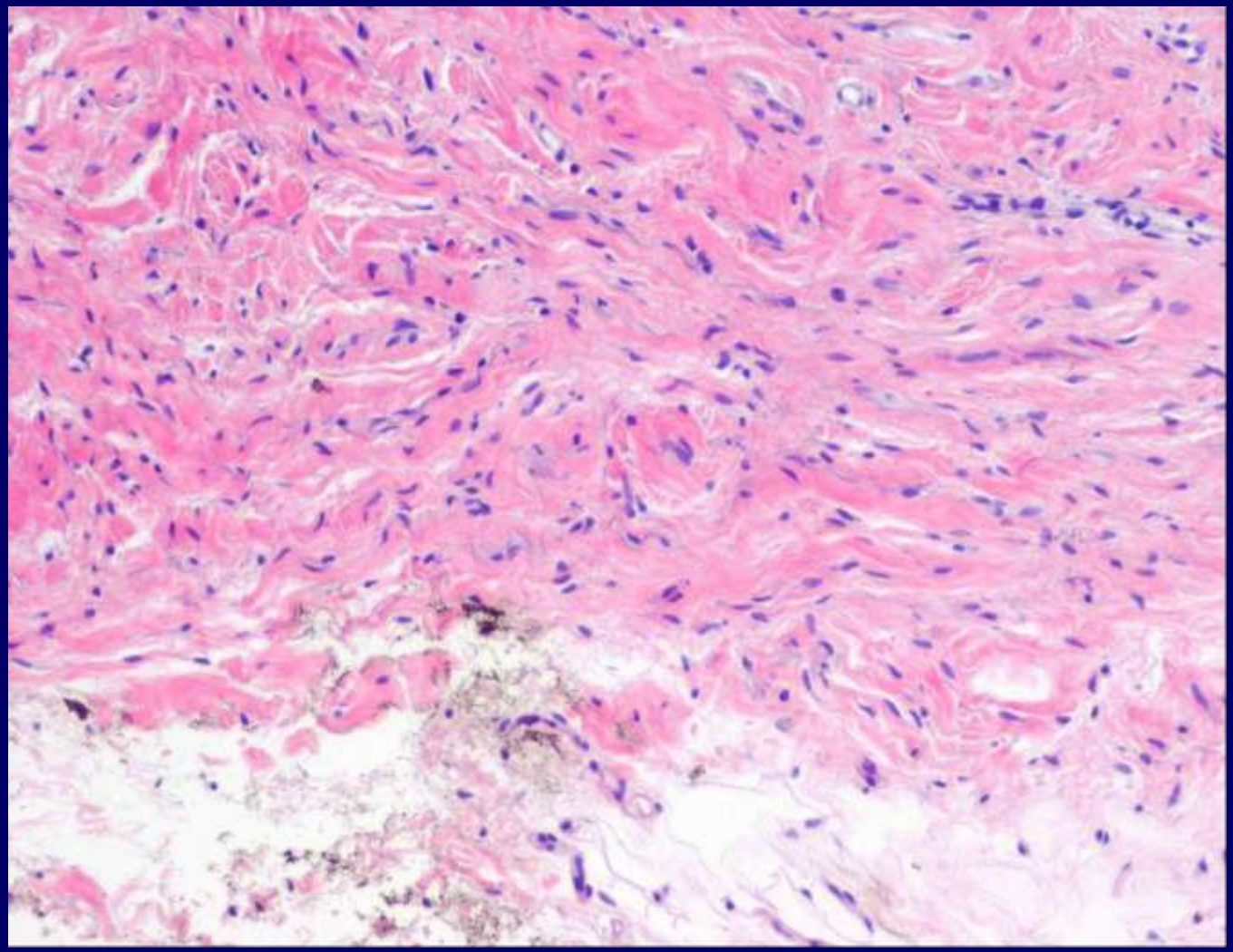
PSEUDOPORPHYRIA

- Patients with clinical and histologic features similar to PCT without abnormal porphyrin levels.
- The disorder may develop in association with medications including:
  - furosemide,
  - nalidixic acid,
  - tetracycline,
  - naproxen,
  - pyroxidine,
  - amiodarone,
  - excess use of UVA tanning beds.
- Clinical features are those of PCT; however, most patients do not manifest hypertrichosis or sclerodermoid plaques.
- H.P.: Identical to that of PCT, with less bl.v wall thickening

PCT. TENSE BULLAE, EROSIONS, AND CRUSTS OF THE DORSAL HANDS.

FIBROTIC UREMIC DERMOPATHY (FUD), NEPHROGENIC SYSTEMIC FIBROSIS (NSF)

- Woody indurated plaques on the extremities > trunk,
- often becoming confluent
- limiting motion of the joints.
- Peau d'orange appearance.
- Systemic affection: Systemic fibrosis, Hypercoagulable states,
- Surgical procedures or administration of IV gadolinium often precede the onset of NFD.



DENSE DERMAL FIBROSIS WITH A PROLIFERATION OF FIBROBLASTS WHICH EXTENDS TO THE DERMAL-SUBCUTANEOUS JUNCTION. SPARSE PERIVASCULAR LYMPHOCYTIC INFILTRATE, INCREASED DERMAL MUCIN WAS DEMONSTRATED BY COLLOIDAL IRON STAIN .

MANAGEMENT

- Plasmapheresis,
- IVIG
- thalidomide,
- UVA with or without psoralen,
- extracorporeal photochemotherapy,
- cyclosporine
- cyclophosphamide

NEPHROGENIC SYSTEMIC FIBROSIS (NEPHROGENIC FIBROSING DERMOPATHY) THIS PATIENT DEMONSTRATES THE PEAU D'ORANGE APPEARANCE OF THE SKIN. IN ADDITION, HE HAD SEVERE RESTRICTION OF THE MOVEMENT OF HIS HANDS, WRISTS AND FEET .

من أقوال زعماء العالم عن الثورة المصرية

- الرئيس الأمريكي باراك أوباما :

"يجب أن نربي أبناءنا ليصبحوا كشباب مصر"(2011/2/14).

سيلفيو برلسكوني رئيس وزراء إيطاليا:

"لا جديد في مصر فقد صنع المصـريون التاريخ كالعادة" (2011/2/15).

رئيس وزراء بريطانيا :

" يجب أن ندرس الثورة المصريه فى مدارسنا." (2011/2/14).

- هاينز فيشر رئيس النمسا :
- "شعب مصر أعظم شعوب الأرض و يستحق جائزة نوبل للسلام"(2011/2/15).
- ستولتنبرج رئيس وزراء النرويج :
- "اليوم كلنا مصريون"(2011/2/15).

Thank You